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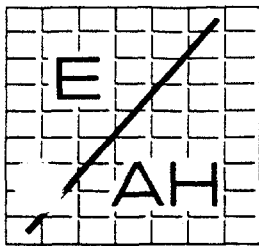
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MEMORANDUM

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FROM: Brian Mulhearn, E/A&H

DATE: June 12, 1996

RE: SWMU 9 Sewage Lagoons, Preliminary Fish Tissue Sample Results; NSA Memphis RFI; CTO - 106

1.0   Introduction

Sediment data from the Solid Waste Management Unit (SWMU) 9 sewage lagoons at Naval Support Activity (NSA) Memphis, indicated a potential risk to ecological receptors. The bioaccumulation potential by demersal (bottom) species such as catfish may indicate a risk to humans from ingestion of fish tissue. The Navy prohibited fishing because the ponds were formerly sewage lagoons until risk management decisions are made by the NSA Memphis BRAC Cleanup Team.

From March 25 to 28, 1996, EnSafe/Allen and Hoshall (E/A&H) conducted a fish study at the SWMU 9 sewage lagoons. For cost-effectiveness, trotlines were the chosen sampling device. The small lagoon (approximately 141,000 square feet) was identified in the study as Pond 1, the large lagoon (approximately 400,000 square feet) was identified as Pond 2. One trotline was set in Pond 1 and two trotlines were set in Pond 2. The number of fish caught for each day in each pond for the duration of the study is presented in Table 1. Only one species of fish, the black bullhead catfish *Ictalurus melas,* was caught during the study.

One composite sample was collected from Pond 1 and two from Pond 2. The sample identification number for Pond 1 was 009J010001, and the identification numbers for Pond 2 were 009J020001 and 009J020002. Two composite samples were collected from Pond 2 based on its size and sediment data. Analytical results from the Ceimic Corporation show concentrations of contaminants in fish tissue above U.S. Environmental Protection Agency (USEPA) Risk Based Concentrations (RBCs) for human health.

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Table 1
Record of Daily Fish Catch

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Date	Species	Number of Fish Pond 1	Number of Fish Pond 2
3/26/96	*Ictalurus melas*	3 medium 1 to 1.5 lb 2 small ¼ lb	2 small ¼ lb
3/27/96	*Ictalurus melas*	2 small ¼ lb	1 small ¼ lb
3/28/96	*Ictalurus melas*	No sample	1 medium 1 lb

2.0   Human Health Risk

Several chemicals were reported in fish tissue collected from Ponds 1 and 2 at NSA Memphis, and screening comparisons were made to evaluate the need for more detailed risk evaluations. Risk estimates presented in this assessment are *preliminary*. Four significant areas of uncertainty exist, which could result in either over- or underestimated exposure assumptions:

1)   The exposure pathway may not be completed in the future (i.e., fishing is prohibited).

2)   No background data are available for comparison to the reported concentrations.

3)   Whole fish were analyzed, and the identified chemicals of potential concern (COPCs) would accumulate more in the liver and skin than in filets.

4)   Suspected sources have not been identified for the COPCs.

Whole fish were analyzed for Target Analyte List (TAL) metals, semivolatile organic compounds (SVOCs), and pesticides/polychlorinated biphenyls (PCBs). Each pond was evaluated separately in this document. As shown in Table 2, all chemicals reported in fish tissue were compared to the applicable USEPA Region III RBCs (USEPA, 1994a; USEPA, 1995b). Based on the tissue data, chemical concentrations greater than the corresponding RBCs were referred to as COPCs. Several exceedances were noted, and risk estimates were developed for all COPCs in accordance with *Risk Assessment Guidance for Superfund* (RAGS) (USEPA, 1989b, 1991a, 1991b, 1992a, 1992b, 1993a, 1993b, 1993c, 1994b, and 1994c) and various supplemental guidance documents specific to USEPA Region IV (USEPA, 1995b).

2.1   Exposure Pathways, Potential Receptors, and Exposure Scenarios

Risk was estimated for two exposure scenarios: subsistence fishing and infrequent fishing. As noted earlier, Ponds 1 and 2 are currently restricted and fishing is not allowed. Therefore, the

Table 2
Identification of Chemicals of Potential Concern
˙ssue Collected from Ponds 1 and 2
˙.SA Memphis RFI

Parameter	009J020001	009J020002	009J010001	Max - 2	RBC	n/c	units	Notes	
Di-n-Butylphthalate		490 J	600 J	490	140000	n	ppb		
4,4'-DDE	42	27	88	42	9.3	c	ppb	1	2
Endrin	40	25	49	40	410	n	ppb		
Aroclor-1254	280	180	100	280	0.41	c	ppb	1	2
Aroclor-1260	79 P	38 JP	100 P	79	0.41	c	ppb	1	2
Aluminum	26.2	81.1	7.1 B	81.1	1400	n	ppm		
Barium	74.2 *	117 *	53.8 *	117	95	n	ppm		2
Calcium	65900 *	55600	45000 *	65900			ppm		
Chromium	6.1	4.2	13.7	6.1	1400	n	ppm		
Copper	7.9 E	8.2 E	14.3 E	8.2	54	n	ppm		
Iron	89.3	198	123	198	410	n	ppm		
Lead	0.8	1.3	0.33	1.3			ppm	1	2
Magnesium	1890	1630	1690	1890			ppm		
Manganese	59.4 N*	84.2 N*	54.8 N*	84.2	6.8	n	ppm	1	2
Mercury	0.04 N*		0.56 N*	0.04	0.41	n	ppm		
Nickel	5.4		7.2	5.4	27	n	ppm		
Potassium	12500	9780	16600	12500			ppm		
Selenium	0.77	0.89	0.86	0.89	6.8	n	ppm		
Sodium	6150	4570	6220	6150			ppm		
Vanadium	1 B	1.7 B	0.84 B	1.7	9.5	n	ppm		
Zinc	99.9 E	96.1 E	88.9 E	99.9	410	n	ppm		
Cobalt	0.27 B	0.43 B		0.43	81	n	ppm		
ˆrsenic		0.12 B		0.12	0.0021	c	ppm		2

Notes:

Max - 2	Maximum concentration reported in pond 2 tissue data (samples 009J020001 and 009J020002)
-	Sample 009J010001 was collected from Pond 1
blank spaces	Indicate compound was not reported
NA	Not applicable
RBC	Risk-based concentration for tissue consumption, excerpted from USEPA Region III's October 1995 Screening Tables.
n	RBC based on noncarcinogenic effects
c	RBC based on carcinogenic effects
*	Laboratory duplicate was not within the method-specified control limits
B	Result is less than the reporting limit and greater than the instrument detection limit
E	The serial dilution was not within the method-specified control limits, and the reported value was estimated because of suspected interference
P	The difference between the values of two instrument columns differs by more than 25%
J	Estimated value
N	Spike sample recovery was not within the method-specied control limits
1	Chemical of potential concern in Pond 1
2	Chemical of potential concern in Pond 2
-	Calcium, iron, magnesium, potassium, and sodium were eliminated as essential nutrients

current land use exposure pathway would not be completed, and risk estimates in this document reflect future land use receptors only. Bullhead catfish, *Ictalurus melas*, were the only potentially edible game fish observed in either pond.

Exposure assumptions from USEPA guidance are used to estimate tissue ingestion, and ultimately, uptake of COPCs (USEPA, 1989a, 1993a). Instead of providing one value for each exposure assumption, USEPA provides ranges of exposure which represent various percentiles of the population, such as tissue ingestion rates from 6.5 to 54 g/day. The ingestion rate of 54 g/day is the Reasonable Maximum Exposure (RME) or upper-bound assumption, and the Central Tendency (CT) or median exposure assumption is 6.5 g/day. Both the RME and CT assumptions were included in this assessment to provide a range of risk estimates for each exposure scenario. Consumption of whole fish was assumed in this assessment, because filet data were not available. Concentrations would likely be greater in whole fish as opposed to filets. Tables 3 and 4 present RME and CT exposure assumptions.

As shown in Tables 3 and 4, typical exposure assumptions were included for the subsistence fisherman scenario for both the adult and child. The exposure for the infrequent fishing (site visitor) scenario was estimated assuming an adolescent and a child are the primary receptors (i.e., more conservative than assuming adult exposure).

2.2   Quantification of Exposure

Exposure was estimated by calculating Chronic Daily Intake (CDI) using the exposure assumptions presented in Tables 3 and 4. CDI was estimated separately for RME and CT exposure for both the subsistence fisherman and the infrequent site visitor. In addition, CDI was estimated separately for Pond 1 and Pond 2. CDI for RME and CT are presented in Tables 5 and 6 for Pond 1 and in Tables 7 and 8 for Pond 2, respectively. As shown in Tables 5 through 8, the exposure point concentration was the maximum concentration reported in the corresponding pond.

Equation 1, shown below, was used to estimate exposure to noncarcinogenic chemicals reported in fish tissue:

$$CDI = \frac{C \times IR \times EF \times ED}{BW \times AT} \tag{1}$$

where:

CDI	=	Chronic Daily Intake (mg/kg-day)
C	=	Concentration in tissue (mg/kg)
IR	=	Tissue ingestion rate (kg/day)
EF	=	Exposure Frequency (days/yr)
ED	=	Exposure Duration (yr)
BW	=	Body Weight (kg)
AT	=	Averaging Time (days)

Table 3
Exposure Assumptions
Tissue Collected from Ponds 1 and 2   **RME Assumptions**
NSA Memphis RFI

Parameter	Abbreviation	Resident (subsistence) Adult	Resident (subsistence) Child	Site Visitor Adolescent	Child	Units
Exposure frequency	EF	350	350	52	52	days/yr
Exposure duration	ED	24	6	4	6	yr
Ingestion rate	IR	0.054	0.054	0.054	0.054	kg/day
Slope factor	SFo	Chemical-specific		Chemical-specific		1/(mg/kg-day)
Reference dose	RfDo	Chemical-specific		Chemical-specific		mg/kg-day
Body weight	BW	70	15	45	15	kg
Averaging time (carcinogen)	ATc	25550	25550	25550	25550	days
Averaging time (noncarcinogen)	ATn	8760	2190	1460	2190	days
Lifetime weighted average	LWA	1.40E+01	NA	1.37E+00	NA	(kg-day-yr)/(day-yr-kg)

Notes:

- *USEPA's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure was used to develop the exposure assumptions above (USEPA 1993).*
- *The EF of 52 days per year was selected as a conservative estimate based on 1 day per weekend*

RME   Reasonable maximal exposure

Table 4
Exposure Assumptions
Tissue Collected from Ponds 1 and 2   **CT Assumptions**
NSA Memphis RFI

Parameter	Abbreviation	Resident (subsistence) Adult	Child	Site Visitor Adolescent	Child	Units
Exposure frequency	EF	234	234	52	52	days/yr
Exposure duration	ED	7	2	7	2	yr
Ingestion rate	IR	0.0065	0.0065	0.0065	0.0065	kg/day
Slope factor	SFo	Chemical-specific		Chemical-specific		1/(mg/kg-day)
Reference dose	RfDo	Chemical-specific		Chemical-specific		mg/kg-day
Body weight	BW	70	15	45	15	kg
Averaging time (carcinogen)	ATc	25550	25550	25550	25550	days
Averaging time (noncarcinogen)	ATn	2555	730	2555	730	days
Lifetime weighted average	LWA	3.55E-01	NA	9.76E-02	NA	(kg-day-yr)/(day-yr-kg)

Notes:

- *USEPA's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure was used to develop the exposure assumptions above (USEPA 1993).*
- *0.0065 kg fish tissue per day was obtained from USEPA's Guidance For Assessing Chemical Contaminant Data For Use In Fish Advisories, 1993*
- *The EF of 52 days per year was selected as a conservative estimate based on 1 day per weekend*

CT   Central Tendency Exposure

Table 5
RME Chronic Daily Intake
Tissue Collected from Pond 1
NSA Memphis RFI

Parameter	EPC (mg/kg)	Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
		adult H-CDI (mg/kg-day)	child H-CDI (mg/kg-day)	LWA C-CDI (mg/kg-day)	adolescent H-CDI (mg/kg-day)	child H-CDI (mg/kg-day)	LWA C-CDI (mg/kg-day)
4,4'-DDE	0.088	6.51E-05	3.04E-04	4.84E-05	1.50E-05	4.51E-05	4.73E-06
Aroclor-1254	0.1	7.40E-05	3.45E-04	5.50E-05	1.71E-05	5.13E-05	5.37E-06
Aroclor-1260	0.1	7.40E-05	3.45E-04	5.50E-05	1.71E-05	5.13E-05	5.37E-06
Barium	NA	NA	NA	NA	NA	NA	NA
Lead	0.33	2.44E-04	1.14E-03	1.81E-04	5.64E-05	1.69E-04	1.77E-05
Manganese	54.8	4.05E-02	1.89E-01	3.01E-02	9.37E-03	2.81E-02	2.94E-03
Arsenic	NA	NA	NA	NA	NA	NA	NA

Notes:
    EPC   *Exposure Point Concentration*
    NA   *Not applicable*
    RME   *Reasonable maximal exposure*
    LWA   *Lifetime weighted average*

Table 6
CT Chronic Daily Intake
Tissue Collected from Pond 1
NSA Memphis RFI

Parameter	EPC (mg/kg)	Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
		adult H-CDI (mg/kg-day)	child H-CDI (mg/kg-day)	LWA C-CDI (mg/kg-day)	adolescent H-CDI (mg/kg-day)	child H-CDI (mg/kg-day)	LWA C-CDI (mg/kg-day)
4,4'-DDE	0.088	5.24E-06	2.44E-05	1.22E-06	1.81E-06	5.43E-06	3.36E-07
Aroclor-1254	0.1	5.95E-06	2.78E-05	1.39E-06	2.06E-06	6.17E-06	3.82E-07
Aroclor-1260	0.1	5.95E-06	2.78E-05	1.39E-06	2.06E-06	6.17E-06	3.82E-07
Barium	NA	NA	NA	NA	NA	NA	NA
Lead	0.33	1.96E-05	9.17E-05	4.58E-06	6.79E-06	2.04E-05	1.26E-06
Manganese	54.8	3.26E-03	1.52E-02	7.61E-04	1.13E-03	3.38E-03	2.09E-04
Arsenic	NA	NA	NA	NA	NA	NA	NA

Notes:
EPC   Exposure Point Concentration
NA   Not applicable
CT   Central Tendency Exposure
LWA   Lifetime weighted average

Table 7
RME Chronic Daily Intake
Tissue Collected from Pond 2
NSA Memphis RFI

Parameter	EPC (mg/kg)	Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
		adult H-CDI (mg/kg-day)	child H-CDI (mg/kg-day)	LWA C-CDI (mg/kg-day)	adolescent H-CDI (mg/kg-day)	child H-CDI (mg/kg-day)	LWA C-CDI (mg/kg-day)
4,4'-DDE	0.042	3.11E-05	1.45E-04	2.31E-05	7.18E-06	2.15E-05	2.26E-06
Aroclor-1254	0.28	2.07E-04	9.67E-04	1.54E-04	4.79E-05	1.44E-04	1.50E-05
Aroclor-1260	0.079	5.84E-05	2.73E-04	4.34E-05	1.35E-05	4.05E-05	4.24E-06
Barium	117	8.65E-02	4.04E-01	6.43E-02	2.00E-02	6.00E-02	6.29E-03
Lead	1.3	9.62E-04	4.49E-03	7.14E-04	2.22E-04	6.67E-04	6.98E-05
Manganese	84.2	6.23E-02	2.91E-01	4.63E-02	1.44E-02	4.32E-02	4.52E-03
Arsenic	0.12	8.88E-05	4.14E-04	6.59E-05	2.05E-05	6.15E-05	6.45E-06

Notes:
    EPC *Exposure Point Concentration*
    RME *Reasonable maximal exposure*
    LWA *Lifetime weighted average*

Table 8
CT Chronic Daily Intake
Tissue Collected from Pond 2
NSA Memphis RFI

		Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
Parameter	EPC (mg/kg)	adult H-CDI (mg/kg-day)	child H-CDI (mg/kg-day)	LWA C-CDI (mg/kg-day)	adolescent H-CDI (mg/kg-day)	child H-CDI (mg/kg-day)	LWA C-CDI (mg/kg-day)
4,4'-DDE	0.042	2.50E-06	1.17E-05	5.83E-07	8.64E-07	2.59E-06	1.61E-07
Aroclor-1254	0.28	1.67E-05	7.78E-05	3.89E-06	5.76E-06	1.73E-05	1.07E-06
Aroclor-1260	0.079	4.70E-06	2.19E-05	1.10E-06	1.63E-06	4.88E-06	3.02E-07
Barium	117	6.97E-03	3.25E-02	1.63E-03	2.41E-03	7.22E-03	4.47E-04
Lead	1.3	7.74E-05	3.61E-04	1.81E-05	2.68E-05	8.03E-05	4.97E-06
Manganese	84.2	5.01E-03	2.34E-02	1.17E-03	1.73E-03	5.20E-03	3.22E-04
Arsenic	0.12	7.14E-06	3.33E-05	1.67E-06	2.47E-06	7.41E-06	4.59E-07

Notes:
EPC   *Exposure Point Concentration*
CT   *Central Tendency Exposure*
LWA   *Lifetime weighted average*

To account for exposure as both a child and an adult, the lifetime weighted average (LWA) was calculated as shown in equation 2, and CDI for carcinogens was estimated as shown in equation 3:

$$LWA = \frac{IR_{child} \times EF_{child} \times ED_{child}}{BW_{child}} + \frac{IR_{adult} \times EF_{adult} \times ED_{adult}}{BW_{adult}} \quad (2)$$

$$CDI = \frac{C \times LWA}{AT} \quad (3)$$

2.3   Toxicological Assessment

USEPA has established a classification system for rating the potential carcinogenicity of environmental contaminants based on the weight of scientific evidence. The cancer classes are described below. Cancer weight-of-evidence class "A" (human carcinogens) means that human toxicological data have shown a proven correlation between exposure and the onset of cancer (in varying forms).   The "B1" classification indicates some human exposure studies have implicated the compound as a probable carcinogen. Weight-of-evidence class "B2" indicates a possible human carcinogen, based on positive laboratory animal data (for carcinogenicity) in the absence of human data. Weight-of-evidence class "C" identifies possible human carcinogens, and class "D" indicates a compound not classifiable with respect to its carcinogenic potential. USEPA has established slope factors (SF) for carcinogenic compounds. The SF is defined as a "plausible upper-bound estimate of the probability of a response (cancer) per unit intake of a chemical over a lifetime" (USEPA, 1989a).

Most substances also can produce noncarcinogenic toxic responses at doses greater than experimentally derived threshold concentrations. USEPA has derived Reference Dose (RfD) values for these substances. A chronic RfD is defined as "an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure concentration for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime."

SFs and RfDs are used in risk calculations to assess the upper-bound level of cancer risk and noncancer hazard associated with exposure to a given concentration of contamination. Toxicological data, including SFs, RfDs, and a general synopsis of the toxic effects of each chemical, are summarized in the following paragraphs related to each COPC.

Arsenic exposure via the ingestion route causes darkening and hardening of the skin in chronically exposed humans. Inhalation exposure to arsenic causes neurological deficits, anemia, and cardiovascular effects (Klaassen, et al., 1986). USEPA set 0.3 μg/kg-day as the RfD for arsenic based on a no observed adverse effect level of 0.8 μg/kg-day in a human exposure study. Arsenic's effects on the nervous and cardiovascular systems are primarily associated with acute exposure to higher levels. Exposure to arsenic-containing materials has been shown to cause cancer in humans. Inhalation of these materials can lead to increased lung cancer risk, and

ingestion of these materials is associated with increased skin cancer rates. Arsenic has been classified as a group A carcinogen by USEPA, which set the 1.5 (mg/kg-day)$^{-1}$ SF for arsenic. As listed in the Integrated Risk Information System (IRIS) (search date 9/1/95), the classification is based on sufficient evidence from human data. Increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic. Human milk contains about 3 μg/L arsenic (Klaassen et al., 1986). The RBC for arsenic in tap water is 0.038 μg/L. As listed in IRIS (search date 9/1/95), the critical effect of this chemical is hyperpigmentation, keratosis, and possible vascular complications. The uncertainty factor was determined to be 3 and the modifying factor was determined to be one.

Barium is used in various alloys, paints, soap, and manufacturing processes. Barium sulfate is used to aid X-ray diagnosis. This element is relatively abundant in nature and is found in plant and animal tissue. Brazil nuts contain 3 to 4 mg per gram nuts. The fatal absorbed dose of barium is approximately 1000 mg (for humans). Major toxic effects of this element are muscle stimulation, central nervous system effects, and effects on the heart (Dreisbach et al., 1987) (Klaassen et al., 1986). USEPA determined the oral and inhalation RfDs to be 0.07 and 1.43E-4 mg/kg-day, respectively.

Manganese is an essential nutrient, but chronic exposure (0.8 mg/kg-day) causes mental disturbances. Studies have shown that manganese uptake from water is greater than manganese uptake from food, and the elderly appear to be more sensitive than children (Klaassen, et al., 1986 Dreisbach et al., 1987). Because of the different uptake rates in water and food, USEPA set two oral RfDs — one for water and one for food. These RfDs are 0.005 and 0.14 mg/kg-day, respectively. Inhalation of manganese dust causes neurological effects and increased incidence of pneumonia. An inhalation RfD was set to 0.0000143 mg/kg-day. According to USEPA, manganese cannot be classified as to its carcinogenicity. Therefore, the cancer class for manganese is group D. As listed in IRIS (search date 6/29/95), this classification is based on existing studies that are inadequate to assess the carcinogenicity of manganese. Manganese is an element considered essential to human health. The typical vitamin supplement dose of manganese is 2.5 mg/day. As listed in IRIS (search date 6/29/95), the critical effects of this chemical in water in the oral summary are Central Nervous System (CNS) effects. The uncertainty factor was determined to be one and the modifying factor was determined to be one. The critical effects of this chemical in food in the oral summary are CNS effects. The uncertainty factor was one and the modifying factor was one. As listed in IRIS (search date 6/29/95), the critical affect of this chemical in the inhalation summary is impairment of neuro-behavioral function. The uncertainty factor was 1,000 and the modifying factor was 1. The IRIS reference concentration (RfC) is 0.00005 mg/m^3.

Lead has been classified as a group B2 carcinogen by USEPA based on animal data. No RfD or SF has been set by USEPA. However, an action level for soil protective of child residents has been proposed by USEPA Region IV, 400 mg/kg. USEPA's Office of Solid Waste and

Emergency Response (OSWER) has recommended a 1,000 mg/kg cleanup standard for industrial properties. USEPA's Office of Water has established a treatment technique action level of 15 μg/L (USEPA, 1994b). As listed in IRIS (search date 10/17/95), this classification is based on sufficient animal evidence. One mouse assay and ten rat bioassays have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short-term studies show that lead affects gene expression. Human evidence is inadequate. An RfD and SF have not been set because of the confounding nature of lead toxicity. Lead can accumulate in bone marrow, and effects have been observed in the CNS, blood, and mental development of children. RfDs are based on the assumption that a threshold must be exceeded to result in toxic effects (other than carcinogenicity). Once lead accumulates in the body, other influences cause the actual levels in the blood to fluctuate — sometimes the lead is attached to binding sites; sometimes lead is free flowing. This fluctuation and lack of previous lead exposure data are two of the reasons lead effects are difficult to predict (Klaassen et al., 1986).

PCB Aroclors are a group of chlorinated hydrocarbons (such as *Aroclors 1254* and *1260*) that accumulate in fat tissue. Occupational exposure (both inhalation and dermal) to PCBs causes eye and lung irritation, loss of appetite, liver enlargement, increased serum liver enzyme levels, rashes and chloracne, and decreased birth weight of infants in heavily exposed worker/mothers. Of the effects listed above, the liver is the primary target organ (Klaassen et al., 1986; Dreisbach et al., 1987). USEPA classified PCB Aroclors as group B2 carcinogens, primarily based on animal data. As listed in IRIS (search date 6/29/95), the basis for the classification is hepatocellular carcinomas in three strains of rats and two strains of mice and inadequate yet suggestive evidence of excess risk of liver cancer in humans by ingestion and inhalation or dermal contact. Oral ingestion of PCBs causes liver and stomach tumors in rat studies. USEPA set 7.7 (mg/kg-day)[-1] as the oral SF for PCB Aroclors, and the RfD for Aroclor 1254 was set to 0.00007 mg/kg-day.

4,4'-DDE is a compound typical of halobenzene derivatives and is a by-product of the pesticide DDT. It is soluble in fat, but not in water, and its primary target organs are the liver and brain. DDE is the form of DDT which accumulates in organisms and is thought to be responsible for egg shell thinning and other ecological effects. DDE bioconcentrates in aquatic organisms and can significantly alter the ecology of some areas, especially where DDE containing aquatic species are a critical species in the food chain. (Dreisbach et al., 1987; Harte et al., 1991). This compound is listed as a B2 carcinogen, and USEPA set the oral SF for DDE to 0.34 (mg/kg-day)[-1].

4,4'-DDT is a pesticide which is soluble in fat, but not in water. The primary target organ of DDT is the brain. Other DDT effects could include cell death in the liver, fatty change of heart muscles, and kidney damage. In a study mentioned in Dreisbach, et al, workers historically exposed to DDT had up to 648 ppm DDT in their body fat, and no adverse health effects were observed. If an individual loses body fat, DDD concentrations are not stored at sufficient

concentrations to induce toxic effects (Dreisbach et al., 1987). As listed in IRIS (search date 1/15/96), the critical noncarcinogenic effect of DDT is liver lesions. USEPA determined the oral RfD to be 0.0005 mg/kg-day, with an uncertainty factor of 100 and a modifying factor of 1.0. Confidence in the RfD is medium. DDT is a class B2 carcinogen based on tumors observed in seven studies in various mouse strains and three studies in rats. DDT is structurally similar to other probable carcinogens, such as DDD and DDE. USEPA determined the slope factor to be 0.34 $(\text{mg/kg-day})^{-1}$.

2.4 Risk Characterization

After calculating CDI, risk estimates were calculated using the RfD or the SF for noncarcinogens and carcinogens, respectively. In accordance with RAGS, the reference dose was used to calculate the hazard quotient (HQ) for each COPC identified in this assessment, as shown in equation 4, and the hazard index (HI) was calculated for each exposure scenario as shown in equation 5:

$$HQ \quad = \quad \frac{CDI}{RfD} \tag{4}$$

$$HI \quad = \quad HQ_1 + HQ_2 + HQ_i... \tag{5}$$

Carcinogenic risk estimates were calculated in accordance with RAGS for each COPC as shown in equation 6. The sum of the risks is referred to as the Incremental excess Lifetime Cancer Risk (ILCR), which was calculated as shown in equation 7:

$$Risk \quad = \quad CDI \times SF \tag{6}$$

$$ILCR \quad = \quad Risk_1 + Risk_2 + Risk_i... \tag{7}$$

Pond 1 risk and hazard estimates for RME and CT exposure are presented in Tables 9 and 10, respectively. Risk and hazard estimates for RME and CT exposure in Pond 2 are presented in Tables 11 and 12, respectively. Risk and hazard estimates are discussed below in terms of ranges. CT results are presented with RME estimates. For example, the CT ILCR (subsistence fisherman) for Pond 1 was estimated to be 2E-5 and the RME ILCR was estimated to be 9E-4. ILCR for the Pond 1 subsistence fisherman is presented as 2E-5 to 9E-4.

2.4.1 Characterization of Pond 1
Subsistence Fisherman
The LWA ILCR was estimated to be 2E-5 to 9E-4 for this scenario. HIs for the adult and child were estimated to be 0.3 to 4 and 1 to 19, respectively.

Infrequent Site Visitor
The LWA ILCR was estimated to be 6E-6 to 8E-5 for this scenario. HIs for the adult and child were estimated to be 0.1 to 1 and 0.3 to 3, respectively.

Table 9
RME Risk Estimates for Tissue Ingestion
Tissue Collected from Pond 1
NSA Memphis RFI

Parameter	RfDo (mg/kg-day)	SFo 1/(mg/kg-day)	Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
			adult HQ	child HQ	LWA ILCR	adolescent HQ	child HQ	LWA ILCR
4,4'-DDE	NA	0.34			1.64E-05			1.61E-06
Aroclor-1254	2E-05	7.7	3.7	17.3	4.23E-04	0.9	2.6	4.14E-05
Aroclor-1260	NA	7.7			4.23E-04			4.14E-05
Barium	0.07	NA						
Lead	NA	NA						
Manganese	0.14	NA	0.3	1.4		0.07	0.20	
Arsenic	0.0003	1.5						
SUM HI			*4*	*19*		*1*	*3*	
SUM ILCR					*9E-04*			*8E-05*

Notes:
RfDo	Oral reference dose
SFo	Oral slope factor
HQ	Hazard quotient
HI	Hazard index
ILCR	Incremental excess lifetime cancer risk
RME	Reasonable maximal exposure

Table 10
CT Risk Estimates for Tissue Ingestion
Tissue Collected from Pond 1
NSA Memphis RFI

Parameter	RfDo (mg/kg-day)	SFo 1/(mg/kg-day)	Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
			adult HQ	child HQ	LWA ILCR	adolescent HQ	child HQ	LWA ILCR
4,4'-DDE	NA	0.34			4.16E-07			1.14E-07
Aroclor-1254	2E-05	7.7	0.3	1.4	1.07E-05	0.10	0.3	2.94E-06
Aroclor-1260	NA	7.7			1.07E-05			2.94E-06
Barium	0.07	NA						
Lead	NA	NA						
Manganese	0.14	NA	0.023	0.11		0.008	0.02	
Arsenic	0.0003	1.5						
		SUM HI	*0.3*	*1*		*0.1*	*0.3*	
		SUM ILCR			*2E-05*			*6E-06*

Notes:
  RfDo   Oral reference dose
  SFo   Oral slope factor
  HQ   Hazard quotient
  HI   Hazard index
  ILCR   Incremental excess lifetime cancer risk
  CT   Central Tendency Exposure

Table 11
RME Risk Estimates for Tissue Ingestion
Tissue Collected from Pond 2
NSA Memphis RFI

Parameter	RfDo (mg/kg-day)	SFo 1/(mg/kg-day)	Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
			adult HQ	child HQ	LWA ILCR	adolescent HQ	child HQ	LWA ILCR
4,4'-DDE	NA	0.34			7.85E-06			7.67E-07
Aroclor-1254	2E-05	7.7	10.4	48.3	1.18E-03	2.4	7.2	1.16E-04
Aroclor-1260	NA	7.7			3.34E-04			3.27E-05
Barium	0.07	NA	1.2	5.8		0.3	0.9	
Lead	NA	NA						
Manganese	0.14	NA	0.4	2.1		0.10	0.31	
Arsenic	0.0003	1.5	0.3	1.4	9.89E-05	0.07	0.21	9.67E-06
SUM HI			*12*	*58*		*3*	*9*	
SUM ILCR					*2E-03*			*2E-04*

Notes:
RfDo   Oral reference dose
SFo   Oral slope factor
HQ   Hazard quotient
HI   Hazard index
ILCR   Incremental excess lifetime cancer risk
RME   Reasonable maximal exposure

Table 12
CT Risk Estimates for Tissue Ingestion
Tissue Collected from Pond 2
NSA Memphis RFI

Parameter	RfDo (mg/kg-day)	SFo 1/(mg/kg-day)	Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
			adult HQ	child HQ	LWA ILCR	adolescent HQ	child HQ	LWA ILCR
4,4'-DDE	NA	0.34			1.98E-07			5.46E-08
Aroclor-1254	2E-05	7.7	0.8	3.9	2.99E-05	0.3	0.9	8.24E-06
Aroclor-1260	NA	7.7			8.45E-06			2.32E-06
Barium	0.07	NA	0.10	0.5		0.034	0.10	
Lead	NA	NA						
Manganese	0.14	NA	0.036	0.2		0.012	0.04	
Arsenic	0.0003	1.5	0.024	0.11	2.50E-06	0.0082	0.02	6.88E-07
SUM HI			*1*	*5*		*0.3*	*1*	
SUM ILCR					*4E-05*			*1E-05*

Notes:
RfDo   *Oral reference dose*
SFo   *Oral slope factor*
HQ   *Hazard quotient*
HI   *Hazard index*
ILCR   *Incremental excess lifetime cancer risk*
CT   *Central Tendency Exposure*

Primary Contributors to Risk and Hazard in Pond 1

In Pond 1 tissue, the primary contributors to risk were: DDE, PCB Aroclor 1254, and PCB Aroclor 1260. The primary contributor to the HI was PCB Aroclor 1254, and manganese contributed a lesser amount. Manganese reported in Pond 1 tissue could be a result of background inorganic concentrations in tissue. However, no background tissue data are currently available.

2.4.2 Characterization of Pond 2

Subsistence Fisherman

The LWA ILCR was estimated to be 4E-5 to 2E-3 for this scenario. HIs for the adult and child were estimated to be 1 to 12 and 5 to 58, respectively.

Infrequent Site Visitor

The LWA ILCR was estimated to be 6E-6 to 8E-5 for this scenario. HIs for the adult and child were estimated to be 0.3 to 3 and 1 to 9, respectively.

Primary Contributors to Risk and Hazard in Pond 2

In Pond 2 tissue, the primary contributors to risk were: PCB Aroclor 1254, PCB Aroclor 1260, arsenic, and DDE. The primary contributor to the HI was PCB Aroclor 1254; barium, manganese, and arsenic contributed a lesser amount. Inorganics reported in tissue could be a result of background inorganic concentrations in tissue. However, no background tissue data are currently available.

Table 13 summarizes risk and hazard estimates for concentrations reported in tissue samples from Ponds 1 and 2.

2.4.3 Discussion

CT estimates were included to present estimate ranges, which indicate marginally elevated risk for the scenarios assessed. RME estimates exceed USEPA's 1E-4 upper-bound acceptable risk range and HI threshold of 1.0. In addition, CT estimates indicate risk and hazard are within an order of magnitude of USEPA's thresholds and do not exceed them.

The primary contributors to risk and hazard in both ponds were PCB Aroclors 1254 and 1260, and DDE. DDE was identified in both Pond 1 and Pond 2 sediment. However, PCBs were not reported in sediment data. DDE and PCBs tend to biomagnify in the food chain, accumulating in lipids and in the liver. Therefore, using linear methods to estimate exposure (such as those typically used in risk assessment) may underestimate exposure. As discussed earlier, exposure may have been under- or overestimated because of several confounding factors.

Because the primary contributors to risk and hazard accumulate in the liver and skin rather than in the filet, exposure estimates could be overestimates. With the exception of one fish sample (see Table 1 for fish sample weights), fish were less than one pound. Fishing in Ponds 1 and 2 is restricted and exposure is limited relative to subsistence fishing. However, risk and hazard estimates for a child and adolescent site visitor were marginally elevated relative to USEPA's

Table 13
Summary of Risk and Hazard Estimates for Ponds 1 and 2
NSA Memphis RFI

Medium	Exposure Pathway	Parameter	Potential Future Resident (subsistence)			Potential Future Infrequent Visitor		
			adult HQ	child HQ	LWA ILCR	adolescent HQ	child HQ	LWA ILCR
Pond 1 Tissue								
	RME Ingestion	4,4'-DDE			1.6E-05			1.6E-06
		Aroclor-1254	3.7	17.3	4.2E-04	0.85	2.6	4.1E-05
		Aroclor-1260			4.2E-04			4.1E-05
		Barium						
		Lead						
		Manganese	0.29	1.35		0.067	0.20	
		Arsenic						
RME Sum Pond 1			*4*	*19*	*9E-04*	*1*	*3*	*8E-05*
	CT Ingestion	4,4'-DDE			4.2E-07			1.1E-07
		Aroclor-1254	0.30	1.39	1.1E-05	0.10	0.31	2.9E-06
		Aroclor-1260			1.1E-05			2.9E-06
		Barium						
		Lead						
		Manganese	0.023	0.11		0.0081	0.024	
		Arsenic						
CT Sum Pond 1			*0.3*	*1*	*2E-05*	*0.1*	*0.3*	*6E-06*
Pond 2 Tissue								
	RME Ingestion	4,4'-DDE			7.8E-06			7.7E-07
		Aroclor-1254	10	48	1.2E-03	2.4	7.2	1.2E-04
		Aroclor-1260			3.3E-04			3.3E-05
		Barium	1.2	5.8		0.29	0.86	
		Lead						
		Manganese	0.44	2.1		0.10	0.31	
		Arsenic	0.30	1.4	9.9E-05	0.068	0.21	9.7E-06
RME Sum Pond 2			*12*	*58*	*2E-03*	*3*	*9*	*2E-04*
	CT Ingestion	4,4'-DDE			2.0E-07			5.5E-08
		Aroclor-1254	0.83	3.9	3.0E-05	0.29	0.86	8.2E-06
		Aroclor-1260			8.4E-06			2.3E-06
		Barium	0.100	0.46		0.034	0.10	
		Lead						
		Manganese	0.036	0.17		0.012	0.037	
		Arsenic	0.024	0.11	2.5E-06	0.0082	0.025	6.9E-07
CT Sum Pond 2			*1*	*5*	*4E-05*	*0.3*	*1*	*1E-05*

Notes:

ND   indicates not determined due to the lack of available risk information.
ILCR   indicates incremental excess lifetime cancer risk
HI   indicates hazard index
RME   reasonable maximum exposure
CT   central tendency exposure

upper-bound thresholds. The primary contributors to risk and hazard were reported in each sample analyzed.

2.4.4 Comparison to FDA Data

U.S. Food and Drug Administration (FDA) action levels and tolerances are established because exposure to poisonous or deleterious substances is sometimes unavoidable and does not represent permissible contaminant concentrations where exposure to contaminants is avoidable. Action levels and tolerances represent limits at or above which FDA will take legal action to remove products from the market. The action levels are established and revised according to criteria specified in Title 21 Code of Federal Regulations, Parts 109 and 509 and are revoked when a regulation establishing a tolerance for the same substance and use becomes effective.

DDE and PCB Aroclors 1254 and 1260 were primary contributors to risk and hazard estimated for tissue ingestion in the technical memorandum. FDA established applicable action levels for DDE and PCBs of 5 mg/kg and 3 mg/kg, respectively. The action level for DDE is based on the edible portion of fish tissue, and the action level for PCBs is based on red meat consumption. An action level for PCBs in fish was not available.

3.0 Ecological Risk

The ecological risk assessment (ERA) is a key component of Baseline Risk Assessments (BRAs). Its purpose is to develop a qualitative and/or quantitative ecological appraisal of the actual or potential effects of contamination identified in the SWMU 9 sewage lagoons. The assessment considers environmental media and exposure pathways that could result in unacceptable levels of exposure to flora and fauna now or in the foreseeable future.

The existing sediment (Table 14) and fish tissue data indicate that a potential risk to ecological receptors exists. Most significant is the presence of PCBs which, although not detected in sediments, were present at elevated levels in the fish tissue. Bioaccumulation of PCBs is therefore assumed to be occurring in fish and may be traveling up the food chain. Measuring the bioaccumulation of toxic substances by aquatic organisms is important in establishing causality for ecological effects, in assessing the health of a community, and providing potential information related to human health risk. Below is a description of stressor characteristics, and their effects on ecological communities.

3.1 Inorganics

In general, heavy metals adversely affect survival, growth, reproduction, development, and metabolism of both terrestrial and aquatic invertebrate species, but effects are substantially modified by physical, chemical, and biological variables. Pascoe et al. (1994) observed that, in general, bioavailability of metals and arsenic in soil to small mammals was limited. Their study also suggests that metal and arsenic intake for higher tropic species may be similarly limited. Most heavy metals do not biomagnify. In contact tests with terrestrial earthworms, the order of toxicity for heavy metals from most toxic to least toxic was copper > zinc > nickel = cadmium > lead (Neuhauser, 1986).

Table 14
SWMU 9 Sewage Lagoons
Organic and Inorganic Constituents in Sediments With HQ > 1

Elements	Number of Samples	Number of Detections	Range of Concentrations	Sediment Screening Value	HQ	ECPC
Organics (ppb) Small Lagoon						
DDD	6	1	9.3	3.3	3	Yes
DDE	6	1	20	3.3	6	Yes
DDT	6	1	4	3.3	1	Yes
Dieldrin	6	1	6.3	3.3	2	Yes
Organics (ppb) Large Lagoon						
DDE	10	1	73	3.3	22	Yes
Inorganics (ppm) Small Lagoon						
Arsenic	6	1	13.1	7.24	2	Yes
Copper	6	3	35.9 - 48.5	18.1	3	Yes
Mercury	6	1	0.13	0.13	1	Yes
Nickel	6	1	23.6	15.9	2	Yes
Inorganics (ppm) Large Lagoon						
Arsenic	10	2	8.3 - 12.5	7.24	2	Yes
Cadmium	10	1	2.6	1	2	Yes
Chromium	10	1	57.1	52.3	1	Yes
Copper	10	8	33.8 - 162	18.7	9	Yes
Lead	10	2	41.2 - 45.7	30.2	2	Yes
Mercury	10	9	0.36 - 2.1	0.13	2	Yes
Nickel	10	4	16.7 - 24.2	15.9	2	Yes
Silver	10	7	2.4 - 21.0	2	11	Yes
Zinc	10	4	131.0 - 303.0	124	2	Yes

Notes:

ECPC	=	Ecological Chemical of Potential Concern
HQ	=	Hazard Quotient
ppb	=	parts per billion
ppm	=	parts per million

Arsenic naturally occurs and, with respect to cycling in the environment, is constantly changing. Many inorganic arsenicals are known teratogens and are more toxic than organic arsenicals (Eisler, 1988). Soil biota appear to be capable of tolerating and metabolizing relatively high concentrations (microbiota to 1,600 ppm) of arsenic but adverse effects to aquatic organisms have been reported at concentrations of 19 to 48 ppb in water (Wang et al., 1984). Arsenic in soil does not appear to magnify along the aquatic food chain.

Hexavalent chromium (Cr VI) produces more adverse effects to biota than does the trivalent phase. In clayey sediments, trivalent chromium dominates and benthic invertebrate bioaccumulation is limited (Neff et al., 1978). The solubility and potential bioavailability of waste chromium added to soil through sewage sludge are modified by soil pH and organic complexing substances (James and Bartlett, 1983).

Copper is an essential micronutrient, and therefore, it is readily accumulated by aquatic organisms. It is a broad-spectrum biocide, which may be associated with both acute and chronic toxicity.

In soil, lead concentrates in organic-rich surface horizons in soil (NRCC, 1973). Lead's estimated residence time in soil is about 20 years (Nriagu, 1978). In sediments, lead is primarily found in association with iron and manganese hydroxides and may also form associations with clays and organic matter. Under oxidizing conditions, lead tends to remain tightly bound to sediments, but is released into the water column under reducing conditions. Lead may accumulate to relatively high concentrations in aquatic biota.

Mercury is a known mutagen, teratogen, and carcinogen. It adversely affects reproduction, growth, development, motor coordination, and metabolism. Mercury has a high potential for bioaccumulation and biomagnification, and is slow to depurate. Organomercury compounds produce more adverse effects than inorganic mercury compounds. Inorganic mercury can be biologically transformed to organic mercury compounds.

In natural waters, zinc speciates into the toxic aquo ion, other dissolved chemical species, various inorganic and organic complexes, and is readily transported. Most zinc introduced into aquatic environments is eventually partitioned into the sediments. Reduced conditions enhance zinc's bioavailability.

3.2   Organics

Organochlorine pesticides have been used extensively in the United States since the 1940s. They appear to be ubiquitous in the environment, being found in surface water, sediment, and biological tissues. They are readily absorbed by warm-blooded species and degradatory products are frequently more toxic than the parent form. Food chain biomagnification is usually low, except in some marine mammals. In soil invertebrates, organochlorine pesticides can accumulate to concentrations higher than those in the surrounding soil, and residues may in turn be ingested by birds and other animals feeding on earthworms (Beyer and Gish, 1980). Most environmental

effects studies have been directed at mammals and birds.

PCBs are distributed worldwide with measurable concentrations recorded in fishery and wildlife resources from numerous locations (Eisler, 1987). They are known to bioaccumulate and to biomagnify within the food chain and to elicit biological effects such as death, birth defects, tumors, and a wasting syndrome. In terrestrial environments, PCBs are rapidly metabolized from the soil into the terrestrial food chain (McKee, 1992). Subsoil-dwelling organisms may directly absorb PCBs and may transfer through the food chain to species.

4.0   Conclusions

Risk assessments are tools used by risk managers (such as the NSA Memphis BRAC Cleanup Team) to determine the need for remediation. In accordance with RAGS, risk management decisions are not included in risk assessments. The general conclusions of this assessment are below:

- Although many uncertainties are inherent in the risk assessment process, a few potentially significant sources of uncertainty and variability were outlined in this memorandum. Risk estimates for human health effects presented in this memorandum are generally near or above USEPA's acceptable risk and hazard thresholds. Uncertainty will be detailed in the RFI report for SWMU 9, which will incorporate tissue data into the BRA.

- The primary contributors to risk and hazard identified in both ponds were PCB Aroclors 1254 and 1260, and DDE; these compounds tend to bioconcentrate up the food chain.

- Due to the time requirement for target species selection, calculations, and the potential human health concerns, the ecological risk assessment at SWMU 9 will be fully addressed in the RFI report for SWMU 9.

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